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ART. I.—FACIAL PARALYSIS OF CEREBRAL ORIGIN OCCURRING IN INFANCY.*

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PARALYSIS of the facial nerve alone is not uncommon in adults; it is perhaps less common in children, yet is not very rare. The two cases which I propose to report are, however, in many respects singular, differing from any others which I have seen, and from any of which I have read, except one very briefly referred to by Volkmann in a foot note in the first number of his *Klinische Sammlung Vortraege*, (Ueber Kinderlehmung und paralytische Contracturen), p. 6.

I have seen several cases of hemiplegia after convulsions in infants, wherein the arm and leg were affected more than the face, but these two are the only cases I have met where the face suffered so severely, and the limbs seemed to be nearly or quite unaffected. The first case was seen but once, and a less complete history was obtained than could be wished.

CASE I.—Lizzie H., aged 9 years, was seen at the Boston Dispensary. Her grandmother stated that when she was

* This paper was prepared as a clinical lecture, and subsequently enlarged and read before the Boston Society for Medical Observation.

about one year old she had a series of fits, beginning about 8 A. M. and lasting three hours. The next day, at about the same time, occurred a second attack, not lasting so long, but more severe. During this time, on the second day, the face was suddenly dragged to the left side. It seemed to the grandmother at the time that the whole of the right side was affected. It was a long time after the fits before she could creep again. As soon as the face was drawn thus to one side the fits ceased. She walked at 1 year and 3 months, and talked very early. She has remained in very nearly the same condition since.

Now the face is totally paralysed on the right, being drawn over to the left; the right eye does not fully close, the lids are separated one-eighth inch or more; this was the case at the beginning of the affection. There is no disturbance of the motions of the limbs; she uses her right hand readily, and walks well. The faradic muscular reaction is very much diminished in the muscles of the right side of the face; they react only to very strong currents. The galvanic reaction is also affected, the muscles reacting only slightly, some not at all, with 25 cells.

CASE II.—Minnie B., aged 17, was also seen at the Dispensary. At about 18 months of age she had a fit; she shivered, stamped, screamed, for about five minutes. Immediately after her mother noticed that her face was drawn to the right, and the left eye seemed larger than the right. For about fifteen minutes she was kept quiet; after that she ran around as well as ever; there was no trouble in the arm. At this time she was cutting both upper eye teeth.

Her catamenia appeared at about 14 years of age; she has been quite irregular, passing over two or three months; there is no dysmenorrhœa nor other disturbance of the function.

Up to two years ago she enjoyed good health without any unusual disturbance except the facial paralysis; then the left arm began to tremble when she used it. This has increased in severity, and during the last few months the right hand has trembled also; for the last year her left leg has trembled. The left leg is weak in walking,—sometimes aches, sometimes feels

dead, as if asleep. The left hand has the same feeling, and the right leg, but not the right hand. The left hand is weaker than the right, compressing the dynamometer to 70, the right hand to 100.

About two years ago she began to have headaches, which lasted three days; then she would be free for a week, when would occur another attack. During these the left side of the head throbbed; there was nausea, the eyes felt heavy, there was diplopia, the sight was blurred. The frequency of these attacks has not increased, but their severity is greater. Just before the attacks she is dizzy, has a funny sensation in her head; feels as if she would drop on the floor without consciousness; feels weak and sleepy after this, and then in about five or ten minutes the headache begins, and always lasts three days. About one year ago she lost consciousness. While the headaches continue the arm and legs feel worse. About the same time at which the headaches began, she noticed that she could not hear with the left ear so well as with the right. Since about one year ago she has had tinnitus.

Now there is total paralysis of the left side of the face. When at rest this is marked, but on any motion of the face it is much more prominent; the left eye is slightly more widely open than the right, and hence looks larger. When closed the left eye only partially shuts, the upper lid falls nearly as low as on the right; its lower border has, however, a slightly wavy outline; the under lid does not rise to meet the upper. The tears are not carried off through the lachrymal canals, and hence the lower part of the eye is frequently suffused with tears. The eyes move to the right well, and can be retained there, but after looking to the extreme left they involuntarily return a little towards the right.

She heard my watch with the right ear at 10 feet or more, with the left ear at 5 feet. The membranes were nearly or quite normal—somewhat concave. The palate and uvula were straight; she tastes on both sides of the tongue.

The measurements of the two sides of the face are of interest; they were taken with calipers, and when there was any possibility of a mistake, were taken several times:

	R.	L.
Occiput to infra-orbital foramen, - -	$5\frac{5}{8}$	$5\frac{8}{8}$
Auditory meatus to " " -	$3\frac{1}{4}$	$3\frac{1}{16}$
Root of nose to zygoma, - - -	$3\frac{10}{16}$	$3\frac{7}{16}$
" " to auditory meatus, -	$4\frac{7}{16}$	$4\frac{6}{16}$
Occiput to external angle of eye, - -	$5\frac{3}{4}$	$5\frac{1}{2}$
Middle upper lip to auditory meatus, -	$4\frac{7}{16}$	$4\frac{3}{16}+$
Length of ear, - - - - - -	$2\frac{1}{8}$	2
Chin to eyebrow, - - - - -	$4\frac{9}{16}$	$4\frac{7}{16}$

It was not easy to measure the circumference of the head accurately on account of the hair; several measurements, however, gave the same result, and pushing nearly all the hair to the left so as to make that side as large as possible, did not materially change the measurement. The circumference around the head at the lower part of the forehead, and just above the ears, gave, on the right, $10\frac{1}{2}$ inches; on the left, 10 inches.

The reaction to electricity was rather unusual. Those muscles about the mouth which are concerned in respiratory movements, and those whose action give the expression of nausea, reacted to a strong faradic current. They responded very feebly to 25 cells of a galvanic battery, though the muscles of mastication lying beneath them acted vigorously, and brought the jaws together forcibly. The muscles about the eye, so far as I could see, did not react to either current. When the electrode was placed over the nerve at its exit from the stylo-mastoid foramen, no muscular reaction was obtained with either current.

The current from 25 cells of a Siemen's and Halske's battery passed from the opposite hand to the face, caused no peculiar sensation except flashes of light—no dizziness.

The temperature taken in the external auditory meatus was 95° on the right, 95.4° on left; the temperature of the room was high—above 70°, and the thermometer would enter only a short distance into the meatus.

The diagnosis of facial paralysis does not satisfy the natural desire to answer the question, "What is the matter with these patients?" Two questions remain,—What is the locality of the lesion? and what is its nature?

In adults it is sometimes difficult to answer these questions; but in these cases there are difficulties which are not found in ordinary cases. As the second case is so much more complete than the other, it will be well to consider that first.

The facial nerve leaves the cavity of the skull with the auditory nerve, enters the fallopian canal, takes a turn almost at right angles directly backwards, is separated from the tympanic cavity only by a thin layer of bone, curves downward and leaves the temporal bone through the stylo-mastoid foramen, and then, or just before its exit commences to curve forwards. It finally divides into branches which are distributed chiefly to the muscles of the face. While in the bony canal branches are given off. At the point where it so turns backwards with such a decided angle, is a collection of nerve cells, the ganglion geniculatum, and from this point arises the superficial petrosal nerve, which forms one of the roots or branches of the spheno-palatine ganglion. At least a part of the motor fibres of the soft palate seem to be included in the superficial petrosal nerve. Next, the facial gives branches to the tympanic plexus, whose action is not well known; also a branch runs to the stapedius muscle, and finally the chorda tympani arises by a recurrent acute angle near the point of exit of the facial from the temporal bone, runs directly upwards, and then curves forwards, crosses the tympanic cavity behind the malleolus, and passes out of the temporal bone and joins the third division of the fifth nerve at an acute angle. The most prominent function of the chorda tympani is to supply the anterior half of the tongue with the sense of taste.

This brief review is useful in recalling to mind the points which are most important for diagnosis. If the facial is paralyzed after giving off the chorda tympani, taste will of course be unaffected; if before that nerve separates, taste will be lost in the corresponding portion of the tongue. If before the stapedius branch is given off, the hearing becomes more acute. If at the point where the petrosal nerve arises the sense of taste will be lost; there will be unnatural acuteness of hearing: the palate will deviate to one side. When the lesion is at the base of the brain, the sense of taste is preserved, but

generally the palate deviates, and there may be acuteness of hearing.

The general rule that when motor nerves are injured in their course, after their exit from their nucleus of origin, the muscles supplied by them lose their reaction to the faradic current, is true also of the facial nerve; hence in all cases of severe lesion of that nerve at the points above mentioned the faradic reaction of the muscles of the face should be lost.

Erb (Ziemssens Handbuch der Speciellen Pathologie und Therapie, Bd. xii., 1 Haelfte, p. 466,) gives other diagnostic signs by which the locality of the lesion may be determined. When there is complete paralysis of the facial branches, paresis of the palate, no disturbance of taste, simple diminution of electric irritability, and when unusual or decussated reflex action is present, lesion of the facial nucleus may be diagnosticated. One will be more certain that the lesion is seated in the bulb when the other nerves arising therefrom are also implicated, (the hypoglossal, accessorius, vagus, trigeminus, abducens.)

Complete paralysis of the facial branches, paresis of the palate, retained reflex action, no disturbance of taste nor hearing, normal electrical irritability, and paralysis of the opposite limbs show that the lesion is in the pons.

Partial paralysis of the facial branches, the upper ones remaining free, paresis of the palate, retained reflex and electrical irritability, and paralysis of limbs on the same side lead to a diagnosis of the location of the lesion above the pons in the crura cerebri and the cerebral hemispheres. Many times a paralysis of the motor oculi of the opposite side aids in fixing the seat of the lesion in the peduncle.

Applying these data for diagnosis to the case above recorded, we find that all the muscles of the face supplied by the facial are paralysed, the eye cannot be entirely closed, this paralysis cannot be called slight, as the attitude of the face is very marked; taste is preserved, and the palate straight. So far as these symptoms enable one to judge, the lesion, according to Erb's statements, is not cerebral. Also the electrical reaction is very much diminished—is indeed wanting for some of the muscles, so far as I could discover by applying the electrode on the skin.

There was no unusual reflex action; other nerves arising from the medulla oblongata were not affected, the only symptom in the least referable to such being that the eyes, when turned to the left, could not be retained in that position, but returned partially to the right. But both eyes were equally affected; had the left abducens been affected in the medulla, the left eye alone would have failed in its motion to the left. There is not sufficient evidence to locate the lesion in the facial nucleus, especially as there seems to be stronger proof in favor of another locality.

The presence of taste and the fact that the palate is not implicated, would lead to a diagnosis of a lesion in that portion of the nerve after the chorda tympani is given off. But the electrical reaction is in part retained; also there are other symptoms which must be considered, especially those which were present when the disease began.

The origin of the paralysis was sudden, after a slight fit. The sudden occurrence might be due to the impression of cold, but the mother remembers no such exposure. Facial paralysis may occur suddenly in a child with otitis, but there would then probably have been such a change of the tympanic cavity that there would be greater deafness than there is in this case, and there is no history of any such inflammation; on the contrary, the child seemed perfectly healthy before the paralysis occurred. If either of these causes had been present, it is scarcely conceivable that there should have been convulsive action sufficiently severe, and lasting long enough for the mother to call it a fit.

It is not uncommon for a child to have a fit and afterwards to be found paralysed on one side, the limbs and face being affected. I have reported two such cases in the *Boston Medical and Surgical Journal*, April 3, 1873. In all such which I have heretofore seen, the arm and leg on one side were paralysed, and partially recovered; in several facial, paralysis occurred on the same side with the paralysis of the limbs, but was not very marked; in some it was scarcely perceptible—in all the eye could be closed fully. In all such cases there were other symptoms which were seen later in life. There was diminished electrical reaction to the faradic current, and, when

tried, to the galvanic current also. At varying intervals after the original attack of convulsions, epileptic fits occurred; one patient at least was brought to me on account of these. In two cases where the facial paralysis was greatest, there was marked atrophy of the paralysed side. Having so recently published cases such as those just referred to, it is unnecessary to report another case at length. In all these cases I think it will be admitted that the disease is seated in the cerebral hemispheres. I have had an opportunity of seeing the brain of one such person, though the clinical history was very imperfect. There was disease of the cerebral hemispheres.

The present case resembles those above referred to, in that the paralysis occurred after convulsions; was hemiplegic; was followed later in life by fits of an epileptic nature, and was succeeded by atrophy, or retarded growth in the parts affected. It differs from them in that the convulsions were very slight, the limbs were little affected, if at all; the facial paralysis was much more marked and more persistent; the upper part of the face was paralysed, so that the eye could not be closed; the electrical reaction suffered more.

The two latter points of difference are the most difficult to reconcile with the supposition of lesion of the hemispheres.

Eulenberg (Lehrbuch der functionelleu Nervenkrankheiten, Berlin, 1871, p. 521,) says that the most valuable conclusions concerning the locality of the disease can be drawn from the greater or less distribution of the paralysis. If all the external facial branches are paralysed, the paralysis is seated not higher than the pons or the facial nucleus. If the upper (frontal or orbicular) branches are entirely intact, and only the muscles of the lower part of the face are paralysed, the locality of the lesion must be sought in a higher region, (cerebral crura, central ganglia, etc.)

Erb declares that when the upper divisions of the facial nerve are paralysed, the lesion is in the pons or below. When the upper division is intact, the lesion is above the pons. Trousseau makes a similar statement;

"However complete may be a hemiplegia depending upon a lesion of the brain, *I have never seen the orbicularis palpebrarum completely paralysed; the eye can always be closed;*

while in the *paralysis of Bell*, the paralysis of the orbicularis palpebrarum is never wanting, and the complete occlusion of the eye is impossible." (The italics are his.) This testimony is strong against the central origin of such a case as the one we are considering.

Within a few years past, however, cases have been reported in which there was paralysis of all the branches of the facial, the eye remaining partially open, and after death lesion of the cerebral hemispheres was found. P. Samt (Zur Pathologie der Rinde, *Archiv. fuer Psychiatrie und Nervenkrankheiten, Bd. V.*, H. 1, p. 201,) reports a case in which there was paralysis of the right side, including the right side of the face, and there was slight paralysis of the orbicularis palpebrarum, and of the corrugator and frontalis on the right. At the autopsy was found a superficial softening of a limited area of the left parietal lobe. The lower part of the parietal lobe was most affected, and next to this the anterior part of the lobus supramarginalis. Here the entire cortex was destroyed, and the medullary substance beneath was attacked. Anteriorly the lesion did not reach the posterior central convolution; posteriorly it passed into the gyrus angularis. At the posterior part of the area the softening did not include the whole depth of the cortex.

The same author reports another case in which all the branches of the facial were paralysed, the lids of the left eye not being entirely shut when the eyes were closed. There was found an aneurism of the right arteria fossae sylvii with hemorrhage into the left temporal lobe, and the right frontal lobe, with hemorrhagic pachymeningitis. (*Berlin klin. Wochenschrift*, 1875, No. 40, p. 542.)

Samt states that he has several times seen the same—slight paralysis of the upper branches of the facial—in cases of disease of the hemispheres. (The note to article in *Archiv. f. Psychiatrie*, Bd. 5, p. 207.)

Carl Stark reports a very interesting case. (*Berlin klin Wochenschrift*, 1874, No. 33.) Marie Mather had left facial paralysis. Feb. 2 she had severe spasm of the whole of the left (paralysed) side of the face, the left half of the forehead, the left eyebrow, strong blepharospasm of the left eye, left

angle of the mouth drawn outwards, also up and down; left ala nasi raised; smacking motions were made with the left half of the lips.* The tongue was at times protruded, and a chewing motion was made. Nearly all the muscles of the face innervated by the facial nerve, and the motor portion of the fifth, were in clonic spasms. There were also clonic spasms of the left hand and left thigh. The clonic spasm of the left facial continued unbroken, except during sleep, for four months.

On each side, over the first frontal convolution, was a rather shallow sac of the arachnoid. A cyst the size of a walnut was found in the upper part of the sulcus praecentralis on the right. The root of the second frontal and lower part of the anterior central convolution were much atrophied from the pressure from the cyst, and were reduced in size at least one-half. When the cyst was emptied the convolutions remained compressed, showing that the pressure had continued for a long time. The convolutions under the first mentioned shallow sacs showed no signs of compression.

Gliky (Zur Pathologie der Grosshiraninde, V. Gliky, *Deutsches Archiv. f. k. Med.*, Bd. XVI., p. 463,) reports an interesting case where there was spasmodic action affecting at first the left arm, with a certain amount of weakness in the limb. Nearly two months later the spasm had extended and involved also the left half of the face, the orbicularis palpebrarum and the muscles of the centre of the face being specially mentioned; the left lower limb was also affected. These spasms were frequently repeated. Not quite three weeks later there was slight paresis of the left half of the face; the left eye was more open than the right. Later this became rather more marked, and there was almost complete paralysis of the left arm and left leg. There was atrophy of the left arm. The galvanic and faradic reaction was retained in all the

*As he states, first, that there was paralysis of the left facial, and then that there was severe spasm of the whole of the paralysed side of the face, and in specifying the muscles affected by the spasm, enumerates those of the forehead, and the eyelids as well as those of the lower part of the face, it seems to me reasonable to conclude that the upper division of the facial was paralysed at the earlier date, though no special mention is made of the condition of the eye.

muscles, only diminished in those of the left arm proportionately to the atrophy. After death that portion of the brain cortex, supposed to contain the centres of motor innervation, was found degenerated, being occupied by a mass of small, round cells, and mentioned as a glioma. The parts affected were the anterior and posterior central convolution, the beginning of the frontal convolutions, part of the supra-marginal convolution, and the lobulus quadratus.

In this case the orbicularis palpebrarum was partially paralysed.

Hitzig reports a case in which there was, temporarily, almost complete paralysis of the entire left facial. After a few minutes the paralysis slowly disappeared, but in the afternoon reappeared for a short time and again disappeared. There was also spasmodic action of the facial muscles. An abcess about 1½ or 2 mm. in diameter, was found just anterior to the lower end of the fissure of Rolando, (Ueber einen interessanten Abscess der Hirnrinde. *Archiv. fuer Psychiatrie und Nervenkrankheiten.* Bd., III., H. 2).

Bernhardt (*Archiv. fuer Psych. u. Nervenkr.* Bd. IV., H. 3, p. 705,) reports a case in which there was great dyspnœa, tendency to sleep, then the left eye was nearly closed, the right eye remained open; there was a tendency to turn the head to the left, the right arm and leg were paralysed; there were found four spots of disease on the left side of the brain, one at the upper part of the anterior central convolution, another in the posterior central convolution, another implicated all the convolutions of the island of Reil; and the fourth, a small part of the lobulus supra-marginalis. Only the cortex was affected in these spots.

The fact that the eye could not be closed, need not then exclude lesion of the cerebral hemisphere.

The fact that the electrical reaction was so very much diminished, would rather exclude lesion of the hemispheres, as in such cases the contractility remains almost indefinitely. Eulenberg says that the faradic and galvanic nervous irritability, as well as the electro-muscular contractility, may remain intact extremely long, and even sometimes be increased. In one such case, of facial paralysis of cerebral origin, he found

the electrical reaction undiminished 18 years after the origin of the affection.

It may be doubted, however, whether the lesion of a nerve so serious as to cause total paralysis for 15 years without the least trace of returning voluntary motion, would leave the muscles in a condition to give any response to electrical irritation.

Also, in cases of hemiplegia of cerebral origin occurring in infancy after convulsions, I have seen in the muscles of the arm as great loss of reaction to electricity, as existed in this case in the facial muscles.

I should not then, from the results of electrical examination alone, feel justified in excluding a central origin of the paralysis. The first case so nearly resembles the second in the diminished reaction, and yet, is so much more evidently of cerebral origin, that it assists in confirming the diagnosis of the second.

The other points of difference are less important. If it is supposed, as I shall suggest soon, that the centre in the cerebral cortex for motor innervation of the face was chiefly affected, it is quite reasonable that in the initial convulsion, the limbs should have been comparatively free from spasmodic action. The mother would naturally call such a convulsion slight, the face being chiefly attacked. The fact that the extent and persistence of the facial paralysis, is so much greater than in the other cases I have seen, may also depend upon the locality of the lesion.

This case resembles the others in that the affection was hemiplegic. Though the child was able to run about immediately after the attack and no *serious* impairment of power was noticed in the arm, yet she has for several years noticed that the left hand was much weaker than the right, and all the unpleasant symptoms were referred to the left side as being the seat of greatest disturbance.

For two years past the patient has had attacks which recurred quite regularly about once a week, beginning with a feeling of dizziness, and a funny sensation in her head; feels as if she would become unconscious, after this feels weak and sleepy, then in about 5 to 10 minutes has a headache which

lasts three days. Each attack is very nearly a repetition of previous ones. Once there was entire loss of consciousness. This is certainly a form of epileptic attack. And in this respect again the symptoms resemble those of the other cases.

Another feature in which this case resembles others of hemiplegia occurring in children after convulsions, is the atrophy of the parts affected. In a patient 14 years old whom I saw some years ago, there had been a series of convulsions, continuing many hours followed by hemiplegia; the leg improved, but the hand and arm did not gain. A short time before I saw her, there was a return of the convulsions. The right arm and leg were less developed than the left, not only in muscular structure, but also in the bones. The measurements were:

	Right.	Left.
Length of the spine of the scapula	4½	4¾
From spine to lower angle	6	6¼
Length of humerus	9½	10½
Length of ulna	8	8½
Circumference of chest at ensiform cart	12	12½
Length of femur	15	16

From lobe of ear over vertex was half inch larger on the left The face looked smaller on the right, but measured the same.*

In one of the cases reported in the Boston *Medical and Surgical Journal* in 1873, the face seemed smaller on the paralysed side. In the present case, the measurements were taken with calipers and parts selected as the points from which to measure where there was the least muscle to cause error. The bones of the face were smaller than on the opposite side.

It is, however, scarcely correct to speak of such cases as atrophy, there is rather arrest of development, and the cause of this is found in the fact that the muscles on the affected side being entirely passive, do not by their act assist the circulation of the blood, indeed, they have less use for blood and receive less. In the face the muscles being pressed

*(As a matter of curiosity I will give the ancestral neuroses in this case: Father is "nervous," mother neuralgic; father's brother died with lung fever and brain trouble, father's sister had paralysis; father's brother's child epileptic at two years of age and again when a young lady, father's sister's child lost its mind, had fits when young and probably, since; father's grandfather found dead in bed.)

against the bones by the traction of the opposite muscles, may be supposed to receive a yet smaller proportion of blood. It is not strange then, that the bones of the paralyzed side should receive less nourishment and should be less developed than those on the opposite side. The atrophy in these cases differs as to cause, from the cases which occur in adults, or older children. In those there is first a disappearance of the adipose tissue, with diminution of the connective tissue. The muscles and bones are affected later. In these cases, as already mentioned, there is rather retarded growth.

One symptom not noticed in any other case, is strongly confirmatory of the location of the lesion in the brain. When the eyes were turned to the left they could not be retained there, but turned back involuntarily towards the right. This defect of power in both eyes could not exist, if the lesion implicated only the left abducens, or its nucleus of origin. That portion of the brain in which the motions of the eyes originates or at least, is co-ordinated, must have been affected, and though we may not be able to exactly locate the region, it is evidently above the nucleus of both the abducens and the third nerve.

Inasmuch, then, as paralysis of the orbicularis palpebrarum, does not necessarily exclude lesion of the hemispheres, as loss of electrical reaction has been seen in other cases of undoubted central origin, and as by the mode of origin—after convulsions, suddenly—as by the present appearance of other hemiplegic symptoms, by the advent of epileptiform attacks and by retarded growth, this case so strikingly resembles other cases following lesion of the hemispheres, and in these respects, differs from cases of simple peripheral lesion, it is proper to diagnosticate central lesion as the cause of the paralysis.

The first case, Lizzie H., so closely resembles the one already considered, that what has been said in regard to the latter will assist to a better understanding of the former; also, the first case so much more closely resembles in the symptoms at its origin, cases of infantile hemiplegia due to cerebral lesion, that its harmony with the second case will serve to give additional probability to the diagnosis already made.

The attack of convulsions was more severe and of longer duration in the first case, and it was repeated the second day. Herein, it resembles other cases of infantile hemiplegia. There was motor disturbance in the limbs, as it was long before the child could creep again, and the grandmother thought the whole right side was affected; in this respect the resemblance to hemiplegia is very close: this soon passed off and when I saw her as in the second case, there was apparently no difference in the facility with which the limbs were used. The total paralysis of the face on the right, coming on suddenly at the end of the fits, the inability to close the right eye, the greatly diminished electrical reaction, were all found in both cases.

It must then be concluded, that the lesion in these two cases, occupies very nearly the same region of the brain, but in the first it was, originally, rather more extensive.

Epileptic attacks have not yet appeared in Lizzie H. These must be expected, and in the future all means of strengthening the nervous system and warding off such attacks, should be resorted to.

The lesion is then not peripheral, but may be situated in the cerebral hemispheres. Can we decide somewhat more accurately as to where it is? We have seen from the cases above referred to, that lesion of certain portions of the cerebral cortex is followed by paralysis of the face, and of the orbicularis palpebrarum. In some of these cases there was spasm preceding, or alternating with the paralysis. The discoveries in regard to the centres of motion in the cerebral cortex for different regions of the body opens an explanation of these cases of paralysis occurring in infancy. The centres for the face and the eye lie near each other, but further from those for the limbs.

Hitzig has drawn the following conclusion from a comparison of cases occurring in human subjects: "Lesions of the upper part of the parietal lobe are accompanied with disturbance of motion in the extremities, lesions of the base of the parietal lobe with motor disturbance of the muscles of the mouth and tongue. If larger areas of the anterior central convolution are affected, more numerous muscular groups are implicated, especially the rest of the facial."

Ferrier has given somewhat more extensive localities to the motor centres, finding that they are situated in "the ascending parietal convolution (posterior central), with its postero-parietal termination as far back as the parieto-occipital fissure, the ascending frontal (anterior central) and posterior termination of the superior frontal convolution." The centres of innervation for the facial muscles, however, are located by Ferrier in nearly the same place as by Hitzig—in the ascending frontal (anterior central) convolution on a level with the posterior termination of the middle frontal and inferior frontal convolutions.

A few autopsies favor the location of the motor center for the facial muscles in man, at the lower portion of the anterior central convolution, as stated by Hitzig and Ferrier.

Though this question as to the location of the motor centres is not yet fully settled, there seems to be more unanimity in regard to the centre for the facial muscles than for those of the limbs.

We may then conclude that the locality given by Hitzig for the centre of innervation of the facial is approximately correct—*i. e.*, at the lower part of the central ascending convolution at about the level of the sulcus praecentralis. We may suppose, then, that the lesion in the cases we are considering is at this locality.

Another interesting question remains to be answered—What is the nature of the lesion in these cases?

I know of the report of but one autopsy in cases similar to those now under consideration, in which a tolerably full account of the symptoms is accompanied with such an account of the changes in the brain as will enable one to form a definite idea of their nature and their locality. The case to which I refer was reported by Thos. Simon in *Berlin klin. Wochenschrift*, 1873, No. 4, 5. It is of sufficient interest, I think, to justify my giving an abstract of it.

Heinrich Pr. was healthy till his second year, towards the end of which he had convulsions, with loss of consciousness, for several hours. When he recovered it was noticed that the right arm and leg were paralysed. Gradually there occurred contraction. Afterwards the boy suffered from epilepsy

every three to five weeks, several attacks recurring in rapid succession. There was no facial paralysis. Sensibility did not seem to be diminished. The right arm and leg were smaller than the left. The epileptic attacks increased in frequency, and the patient died in a state of dementia when about 23 years old. Secondary changes were found in the lateral columns of the cord. The skull was long, unusually thick; the right half of the coronal suture was normal; the left in its upper portion was entirely anchylosed, and the descending portion was not very clearly marked. The sagittal suture in its posterior two-thirds was entirely anchylosed. The pia mater was dull, and adherent to the cortex in many places. On the left hemisphere the convolutions were depressed over a large surface, so that there was evidently a considerable loss of substance. The lower half of the posterior central convolution, the lowermost point of the anterior, the convolutions of the island of Reil, the entire posterior two-thirds of the third frontal convolution, the parietal convolutions with the exception of the upper, and all the occipital convolutions excepting the cuneus and the lobulus quadratus, were united into a firm mass, in which the separate convolutions projected not more than the thickness of a card. On section this mass was seen to consist of a gray translucent tissue, in which were separate small cavities, similar to small centres of softening. The altered convolutions were sharply defined from the healthy. The ventricles were large, especially the posterior cornua. The central ganglia were normal, the left lenticular nucleus contained cavities around the vessels. The convolution of the hippocampus major was implicated in the sclerotic change. Cerebellum and pons were normal. The left pyramid of the medulla was markedly gray and translucent. Only the cortical substance was affected, the white medullary substance having escaped. The gray substance was changed into a net-work of numberless very fine but sharply defined fibres, inextricably woven together, with individual round cells, or oblong and round nuclei, scattered through the mass. Here and there the cells were grouped together in small foci.

The author thinks that the disease had advanced during the latter part of life, and so the results at the autopsy did not

show exactly the parts affected in infancy. Most interesting is the fact that the region assigned by Hitzig to the centre for the facial nerve had escaped.

When a comparison is made between the lesion supposed to exist in such cases as those we have been considering, and that which is found after infantile paralysis, a similarity is seen between the cerebral disease and the spinal. So close a resemblance exists in the symptoms of these two affections, that there is often an error made in diagnosis. In infantile paralysis the motor cells of the anterior cornua of the spinal cord disappear; there is left more or less deformity of the gray substance, with more or less sclerosis. A similar change was found in the gray substance of the brain, according to Simon's autopsy. It is desirable that further investigations should be made in these cases. Unfortunately, however, the patient lives many years after the advent of the disease, dies from some other disease, and the physician who finally attends him has no opportunity to carefully examine the condition of the paralysed parts, and no autopsy is asked. A few autopsies of such cases would assist in clearing up the question of location of the motor centres.

Another interesting subject for study is, whether the lesion of these centres causes a more rapid loss of electrical excitability in the muscles and nerves, than lesion elsewhere in the hemispheres, and whether this influence of lesion of these centres is more marked, when it occurs in infancy, than when it occurs in adult life. Or is the loss of electrical excitability due simply to long disuse, dating from a period when the motor power was, as yet, only imperfectly developed. Certain it is that in all the cases of this nature in which I have made careful, comparative examination, there has been this diminution of response to the faradic current in the muscles.

Another question of interest arises in these cases. When right hemiplegia occurs in infants after convulsions, the child may, subsequently, learn to talk at a later age than usual, there may be some lack of readiness in talking, or there may be no trouble in this respect. I have not yet seen a case of entire inability to talk after the lapse of many years. Is this absence of aphasia owing to the fact that only the motor cen-

tres are affected? Or is the explanation to be found in the fact, that the child not having learned to talk, the right brain takes up a function naturally performed by the left, as in the case of dogs, in which Soltmann found that extirpation of motor centres on one side, caused permanent hemiplegia when they had reached a certain age, but when the experiment was performed on new born pups, before they could walk, no hemiplegia was noticed subsequently.

One class of symptoms in Case II, has not yet been noticed, as they are of later origin and do not affect the diagnosis of the condition at an earlier period. I refer to the changes in sensation and in motor power which began two years ago. These are evidently the results of secondary changes, probably of the nature of sclerosis. About the same time the epileptiform headaches commenced. Is not this a clew to the conditions giving rise to epileptic attack later in life, in other cases of infantile hemiplegia following convulsions of cerebral origin?

ART. II.—NOTE ON THE PHYSIOLOGY OF THE RESPIRATORY APPARATUS.

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IN that classical work (*Bemerkungen ueber die Thaetigkeit der Automatischen Nervencentra inbesondere ueber die Athmenbewegungen*, von Dr. J. Rosenthal, Erlangen, 1875), on the physiology of the respiratory movements, Rosenthal's conclusions were that the movements of respiration were excited by the irritation of the blood upon the nerve centres of respiration. The transmission of this irritation to the muscles and nerves concerned, suffered a resistance through which the continual excitation was changed into a rhythmical action. This resistance is increased through the action of the superior laryngeal and diminished through the pneumogastric. A con-